```
      (FILE 'HOME' ENTERED AT 12:43:40 ON 30 MAR 2007)

      FILE 'REGISTRY' ENTERED AT 12:43:52 ON 30 MAR 2007
L1                STRUCTURE UPLOADED
L2              0 S L1 FAM SAM
L3              3 S L1 FAM FULL

      FILE 'CAPLUS' ENTERED AT 12:44:44 ON 30 MAR 2007
L4             13 S L3
L5              0 S L4 AND ((COMPLEX(W)REGIONAL(W)PAIN(W)SYNDROME)OR(REFLEX(W)SYM
L6              2 S L4 AND PAIN
L7          50526 S TNF(W)ALPHA
L8             16 S L7 AND ((COMPLEX(W)REGIONAL(W)PAIN(W)SYNDROME)OR(REFLEX(W)SYM
L9              3 S L8 NOT PY>2004
L10             8 S L8 AND (INHIBITOR OR ANTAGONIST)
```

=> file registry
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'REGISTRY' ENTERED AT 12:43:52 ON 30 MAR 2007
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Property values tagged with IC are from the ZIC/VINITI data file
provided by InfoChem.

STRUCTURE FILE UPDATES: 29 MAR 2007 HIGHEST RN 928707-03-3
DICTIONARY FILE UPDATES: 29 MAR 2007 HIGHEST RN 928707-03-3

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TSCA INFORMATION NOW CURRENT THROUGH December 2, 2006

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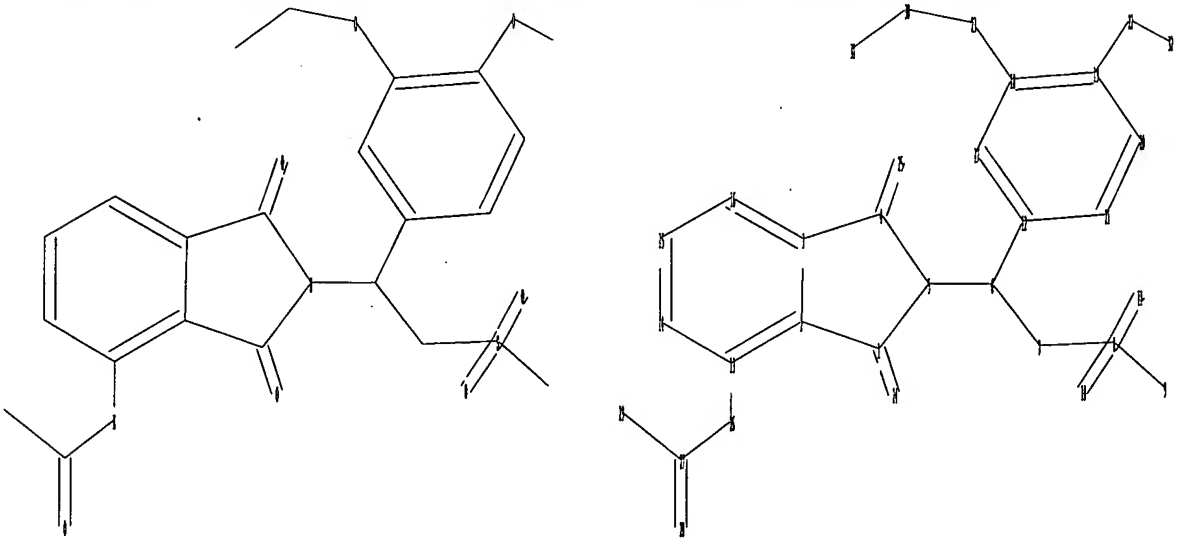
REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document. For information
on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

=>
Uploading C:\Program Files\Stnexp\Queries\10693722specificfinal.str



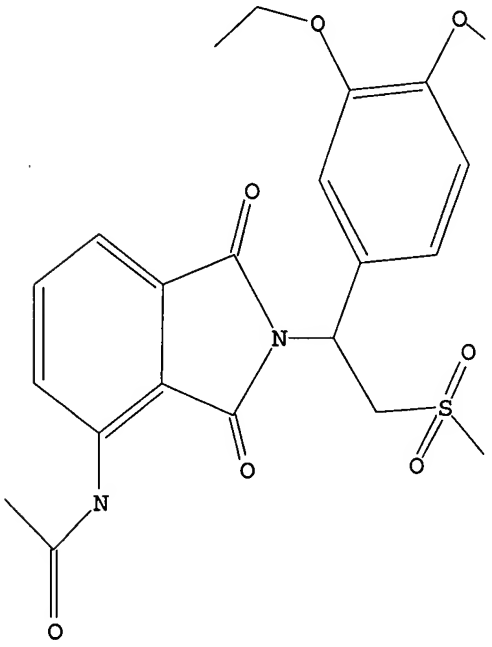
chain nodes :
6 7 8 9 10 11 22 23 24 25 26 27 28 29 30 31 32
ring nodes :
1 2 3 4 5 12 13 14 15 16 17 18 19 20 21
chain bonds :
1-24 4-25 5-6 6-7 6-12 7-8 8-9 8-10 8-11 13-26 18-23 19-22 22-32 23-30
26-27 27-28 27-29 30-31
ring bonds :
1-2 1-5 2-3 2-13 3-4 3-16 4-5 12-17 12-21 13-14 14-15 15-16 17-18 18-19
19-20 20-21

exact/norm bonds :
1-2 1-5 1-24 3-4 4-5 4-25 5-6 7-8 8-9 8-10 8-11 13-26 18-23 19-22
22-32
23-30 26-27 27-28
exact bonds :
6-7 6-12 27-29 30-31
normalized bonds :
2-3 2-13 3-16 12-17 12-21 13-14 14-15 15-16 17-18 18-19 19-20 20-21

Match level :
1:Atom 2:Atom 3:Atom 4:Atom 5:Atom 6:CLASS 7:CLASS 8:CLASS 9:CLASS 10:CLASS
11:CLASS 12:Atom 13:Atom 14:Atom 15:Atom 16:Atom 17:Atom 18:Atom 19:Atom
20:Atom 21:Atom
22:CLASS 23:CLASS 24:CLASS 25:CLASS 26:CLASS 27:CLASS 28:CLASS 29:CLASS
30:CLASS 31:CLASS
32:CLASS

L1 STRUCTURE UPLOADED

=> d l1
L1 HAS NO ANSWERS
L1 STR



Structure attributes must be viewed using STN Express query preparation.

=> s l1 fam sam
SAMPLE SEARCH INITIATED 12:44:16 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED - 2 TO ITERATE

100.0% PROCESSED 2 ITERATIONS 0 ANSWERS
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS: ONLINE **COMPLETE**

BATCH **COMPLETE**
PROJECTED ITERATIONS: 2 TO 124
PROJECTED ANSWERS: 0 TO 0

L2 0 SEA FAM SAM L1

=> s l1 fam full
FULL SEARCH INITIATED 12:44:21 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED - 40 TO ITERATE

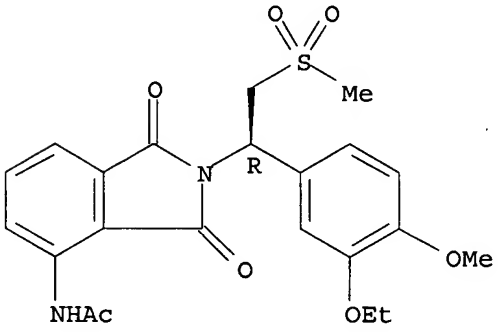
100.0% PROCESSED 40 ITERATIONS 3 ANSWERS
SEARCH TIME: 00.00.01

L3 3 SEA FAM FUL L1

=> d l3 scan

L3 3 ANSWERS REGISTRY COPYRIGHT 2007 ACS on STN
IN Acetamide, N-[2-[(1R)-1-(3-ethoxy-4-methoxyphenyl)-2-(methylsulfonyl)ethyl]-2,3-dihydro-1,3-dioxo-1H-isoindol-4-yl]- (9CI)
MF C22 H24 N2 O7 S

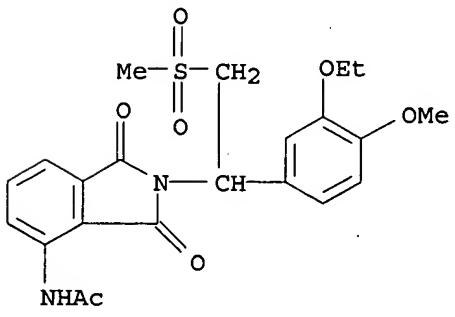
Absolute stereochemistry. Rotation (-).



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):2

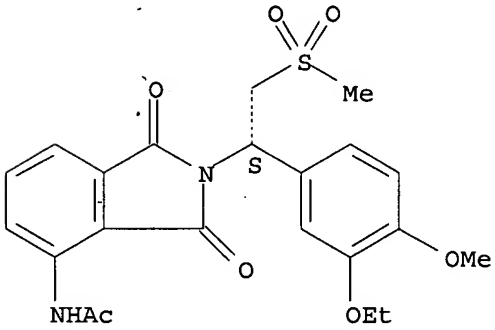
L3 3 ANSWERS REGISTRY COPYRIGHT 2007 ACS on STN
IN Acetamide, N-[2-[1-(3-ethoxy-4-methoxyphenyl)-2-(methylsulfonyl)ethyl]-2,3-dihydro-1,3-dioxo-1H-isoindol-4-yl]- (9CI)
MF C22 H24 N2 O7 S



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

L3 3 ANSWERS REGISTRY COPYRIGHT 2007 ACS on STN
IN Acetamide, N-[2-[(1S)-1-(3-ethoxy-4-methoxyphenyl)-2-(methylsulfonyl)ethyl]-2,3-dihydro-1,3-dioxo-1H-isoindol-4-yl]- (9CI)
MF C22 H24 N2 O7 S

Absolute stereochemistry. Rotation (+).



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

ALL ANSWERS HAVE BEEN SCANNED

=> file caplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	67.70	67.91

FILE 'CAPLUS' ENTERED AT 12:44:44 ON 30 MAR 2007
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
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FILE COVERS 1907 - 30 Mar 2007 VOL 146 ISS 15
FILE LAST UPDATED: 29 Mar 2007 (20070329/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply. They are available for your review at:

http://www.cas.org/infopolicy.html

```
=> s l3
L4            13 L3

=> s l4 and
((complex(w)regional(w)pain(w)syndrome)or(Reflex(w)sympathetic(w)dystrophy))
       1329086 COMPLEX
         67862 REGIONAL
         49424 PAIN
        123930 SYNDROME
           110 COMPLEX(W)REGIONAL(W)PAIN(W)SYNDROME
         25143 REFLEX
         39834 SYMPATHETIC
         13005 DYSTROPHY
           175 REFLEX(W)SYMPATHETIC(W)DYSTROPHY
L5             0 L4 AND ((COMPLEX(W)REGIONAL(W)PAIN(W)SYNDROME)OR(REFLEX(W)SYMPAT
                 HETIC(W)DYSTROPHY))

=> l4 and pain
L4 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s l4 and pain
         49424 PAIN
L6             2 L4 AND PAIN

=> d l6 1-2 ti abs bib

L6   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2007 ACS on STN
TI   Use of (+)-2-[1-(3-ethoxy-4-methoxyphenyl)-2-methylsulfonylethyl]-4-
     acetylaminoisoindoline-1,3-dione and compositions thereof for inhibiting
     TNF-α production and PDE4 activity
GI
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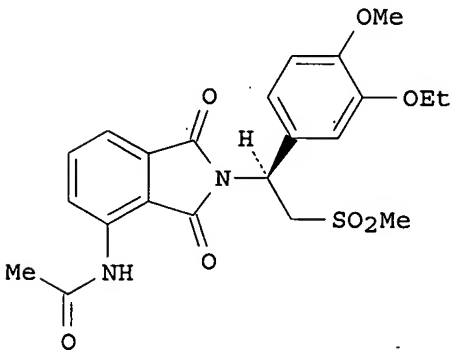


I

AB The invention discloses stereomerically pure (S)-2-[1-(3-Ethoxy-4-methoxyphenyl)-2-methylsulfonylethyl]-4-acetylaminoisoindoline-1,3-dione (+)-I, substantially free of its (-)-isomer, and prodrugs, metabolites, polymorphs, salts, solvates, hydrates, and clathrates thereof. Methods of using and pharmaceutical compns. comprising (+)-I for treating and/or preventing disorders ameliorated by the reduction of levels of tumor necrosis factor α (TNF-α) or the inhibition of phosphodiesterase IV (PDE4) are also disclosed. Examples include the synthesis and resolution of

(+)-I, thirteen bioassays, an aqueous solubility study, and three formulations. For instance, 3-nitrophthalic acid was hydrogenated using 10% Pd/C in EtOH to give the amine (84%), which was condensed with Ac2O to afford 3-acetamidophthalic anhydride (61%). Reaction of the phthalic anhydride with 1-(3-ethoxy-4-methoxyphenyl)-2-(methylsulfonyl)ethylamine to give I (59%), followed by resolution with N-acetyl-L-leucine in MeOH provided (+)-I (90% recovery, 98.4% ee). The latter inhibited LPS-induced TNF-α production by human whole blood and PDE4 activity with IC50 values of 294 nM and 73.5 nM, resp. (+)-I showed >500-fold to >40,000-fold selectivity for PDE4 over PDE1, PDE2, PDE3, PDE5, and PDE6. In addition, (+)-I suppressed LPS-induced lung neutrophilia in conscious ferrets with an ED50 of 0.8 mg/kg. Thus, (+)-I and its pharmaceutical compns. are useful for treating and/or preventing cancer, depression, and a variety of allergic, inflammatory, and autoimmune disorders (no data).

AN 2003:777583 CAPLUS <<LOGINID::20070330>>

DN 139:296870

TI Use of (+)-2-[1-(3-ethoxy-4-methoxyphenyl)-2-methylsulfonylethyl]-4-acetylaminoisoindoline-1,3-dione and compositions thereof for inhibiting TNF-α production and PDE4 activity

IN Schafer, Peter H.; Muller, George W.; Man, Hon-Wah; Ge, Chuansheng

PA Celgene Corporation, USA

SO PCT Int. Appl., 57 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 3

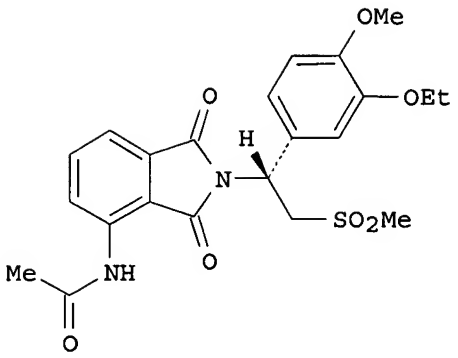
	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003080049	A1	20031002	WO 2003-US8738	20030320
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2479666	A1	20031002	CA 2003-2479666	20030320
	AU 2003224729	A1	20031008	AU 2003-224729	20030320
	EP 1485087	A1	20041215	EP 2003-721414	20030320
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	CN 1652772	A	20050810	CN 2003-811093	20030320
	JP 2005525386	T	20050825	JP 2003-577877	20030320
	NZ 535798	A	20060428	NZ 2003-535798	20030320
PRAI	US 2002-366515P	P	20020320		
	US 2003-438450P	P	20030107		
	WO 2003-US8738	W	20030320		

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN

TI Use of (-)-2-[1-(3-ethoxy-4-methoxyphenyl)-2-methylsulfonylethyl]-4-acetylaminoisoindoline-1,3-dione and compositions thereof for inhibiting TNF-α production and PDE4 activity

GI



I

AB The invention discloses stereomerically pure (R)-2-[1-(3-Ethoxy-4-methoxyphenyl)-2-methylsulfonylethyl]-4-acetylaminoisoindoline-1,3-dione (-)-I, substantially free of its (+)-isomer, and prodrugs, metabolites, polymorphs, salts, solvates, hydrates, and clathrates thereof. Methods of using and pharmaceutical compns. comprising (-)-I for treating and/or preventing disorders ameliorated by the reduction of levels of tumor necrosis factor α (TNF-α) or the inhibition of phosphodiesterase IV (PDE4) are also disclosed. Examples include the synthesis and resolution of (-)-I, seven bioassays, an aqueous solubility study, and three formulations. For instance, 3-nitrophthalic acid was hydrogenated using 10% Pd/C in EtOH to give the amine (84%), which was condensed with Ac2O to afford 3-acetamidophthalic anhydride (61%). Reaction of the phthalic anhydride with 1-(3-ethoxy-4-methoxyphenyl)-2-(methylsulfonyl)ethylamine to give I (59%), followed by resolution with N-acetyl-D-leucine in MeOH provided (-)-I (90% recovery, 98.4% ee). The latter inhibited LPS-induced TNF-α production by human whole blood and PDE4 activity with IC50 values of 371 nM and 611 nM, resp. (-)-I showed >45-fold to >39,000-fold selectivity for PDE4 over PDE1, PDE2, PDE3, PDE5, and PDE6. Thus, (-)-I and its pharmaceutical compns. are useful for treating and/or preventing cancer, depression, and a variety of allergic, inflammatory, and autoimmune disorders (no data).

AN 2003:777582 CAPLUS <<LOGINID::20070330>>
DN 139:296869
TI Use of (-)-2-[1-(3-ethoxy-4-methoxyphenyl)-2-methylsulfonylethyl]-4-acetylaminoisoindoline-1,3-dione and compositions thereof for inhibiting TNF-α production and PDE4 activity
IN Schafer, Peter H.; Muller, George W.; Man, Hon-Wah; Ge, Chuansheng
PA Celgene Corporation, USA
SO PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003080048	A1	20031002	WO 2003-US8737	20030320

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

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     AU 2003222034         A1     20031008     AU 2003-222034          20030320
PRAI US 2002-366516P       P      20020320
     US 2003-438448P       P      20030107
     WO 2003-US8737        W      20030320
RE.CNT  3      THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
               ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> s TNF(w)alpha
         67328 TNF
       1676057 ALPHA
L7       50526 TNF(W)ALPHA

=> s l7 and
((complex(w)regional(w)pain(w)syndrome)or(Reflex(w)sympathetic(w)dystrophy))
       1329086 COMPLEX
         67862 REGIONAL
         49424 PAIN
        123930 SYNDROME
           110 COMPLEX(W)REGIONAL(W)PAIN(W)SYNDROME
         25143 REFLEX
         39834 SYMPATHETIC
         13005 DYSTROPHY
           175 REFLEX(W)SYMPATHETIC(W)DYSTROPHY
L8          16 L7 AND ((COMPLEX(W)REGIONAL(W)PAIN(W)SYNDROME)OR(REFLEX(W)SYMPAT
               HETIC(W)DYSTROPHY))

=> d l8 not py>2004
'NOT' IS NOT A VALID FORMAT FOR FILE 'CAPLUS'
'PY>2004' IS NOT A VALID FORMAT FOR FILE 'CAPLUS'

The following are valid formats:

ABS --------- GI and AB
ALL --------- BIB, AB, IND, RE
APPS -------- AI, PRAI
BIB --------- AN, plus Bibliographic Data and PI table (default)
CAN --------- List of CA abstract numbers without answer numbers
CBIB -------- AN, plus Compressed Bibliographic Data
CLASS ------- IPC, NCL, ECLA, FTERM
DALL -------- ALL, delimited (end of each field identified)
DMAX -------- MAX, delimited for post-processing
FAM --------- AN, PI and PRAI in table, plus Patent Family data
FBIB -------- AN, BIB, plus Patent FAM
IND --------- Indexing data
IPC --------- International Patent Classifications
MAX --------- ALL, plus Patent FAM, RE
PATS -------- PI, SO
SAM --------- CC, SX, TI, ST, IT
SCAN -------- CC, SX, TI, ST, IT (random display, no answer numbers;
              SCAN must be entered on the same line as the DISPLAY,
              e.g., D SCAN or DISPLAY SCAN)
STD --------- BIB, CLASS

IABS -------- ABS, indented with text labels
IALL -------- ALL, indented with text labels
IBIB -------- BIB, indented with text labels
IMAX -------- MAX, indented with text labels
ISTD -------- STD, indented with text labels

OBIB -------- AN, plus Bibliographic Data (original)
OIBIB ------- OBIB, indented with text labels

SBIB -------- BIB, no citations
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SIBIB ------- IBIB, no citations

HIT --------- Fields containing hit terms
HITIND ------ IC, ICA, ICI, NCL, CC and index field (ST and IT)
              containing hit terms
HITRN ------- HIT RN and its text modification
HITSTR ------ HIT RN, its text modification, its CA index name, and
              its structure diagram
HITSEQ ------ HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
FHITSTR ----- First HIT RN, its text modification, its CA index name, and
              its structure diagram
FHITSEQ ----- First HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
KWIC -------- Hit term plus 20 words on either side
OCC --------- Number of occurrence of hit term and field in which it occurs

To display a particular field or fields, enter the display field
codes.  For a list of the display field codes, enter HELP DFIELDS at
an arrow prompt (=>).  Examples of formats include: TI; TI,AU; BIB,ST;
TI,IND; TI,SO.  You may specify the format fields in any order and the
information will be displayed in the same order as the format
specification.

All of the formats (except for SAM, SCAN, HIT, HITIND, HITRN, HITSTR,
FHITSTR, HITSEQ, FHITSEQ, KWIC, and OCC) may be used with DISPLAY ACC
to view a specified Accession Number.
ENTER DISPLAY FORMAT (BIB):ti

L8   ANSWER 1 OF 16  CAPLUS  COPYRIGHT 2007 ACS on STN
TI   Increased endothelin-I and diminished nitric oxide levels in blister
     fluids of patients with intermediate cold type complex
     regional pain syndrome type I


=> s l8 not py>2004
       2909281 PY>2004
L9           3 L8 NOT PY>2004

=> d l9 1-3 ti

L9   ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2007 ACS on STN
TI   Mast cells are involved in inflammatory reactions during Complex
     Regional Pain Syndrome type 1

L9   ANSWER 2 OF 3  CAPLUS  COPYRIGHT 2007 ACS on STN
TI   Evidence for local inflammation in complex regional
     pain syndrome type 1

L9   ANSWER 3 OF 3  CAPLUS  COPYRIGHT 2007 ACS on STN
TI   Increased production of nitric oxide stimulated by interferon-γ from
     peripheral blood monocytes in patients with complex
     regional pain syndrome


=> d l9 1-3 ti abs bib

L9   ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2007 ACS on STN
TI   Mast cells are involved in inflammatory reactions during Complex
     Regional Pain Syndrome type 1
AB   Background: The Complex Regional Pain
     Syndrome type 1 (CRPS1) is a complication of surgery or trauma but
     spontaneous development is also described.  Although the pathogenesis
     remains debatable, afferent, efferent and central nervous system
```

mechanisms are proposed. Recently we showed involvement of the proinflammatory cytokines IL-6 and TNFα which is direct evidence for an inflammatory process. Many types of cells, such as activated T lymphocytes, monocytes,macrophages and skin resident cells like mast cells, could contribute to the production of cytokines. Involvement of mast cells is relatively easy to detect by measurement of tryptase. Aim: To establish whether mast cells are involved in the inflammatory reactions during CRPS1. Methods: Twenty patients fulfilling the Bruehl criteria with CRPS1 in one extremity were studied. Impairment was assessed by registration of pain and measurement of differences in temperature, volume and mobility between the involved and uninvolved extremity. Blisters were made with a suction method in order to determine cytokines and mast cell derived tryptase in the involved and uninvolved extremity. Results: In the blister fluid a significant difference was found between the involved and uninvolved extremity in IL-6 {53.5 (17.3-225) vs. 6.2 (2-20.3) pg/mL}, TNFα {31 (15.5-131.5) vs. 8 (4-39) pg/mL}, and tryptase {37 (20.5-62.3) vs. 12.5 (6.7-23.5) ng/mL}. There was a significant correlation between the intensity of pain and tryptase levels in the involved extremity. Conclusion: Mast cells are involved in inflammatory reactions during the CRPS1. Mast cells could play a role in the production of cytokines such as TNFα .

AN 2004:169482 CAPLUS <<LOGINID::20070330>>
DN 140:337839
TI Mast cells are involved in inflammatory reactions during Complex Regional Pain Syndrome type 1
AU Huygen, Frank J. P. M.; Ramdhani, Navin; van Toorenenbergen, Albert; Klein, Jan; Zijlstra, Freek J.
CS Department of Anaesthesiology, Pain Treatment Center, Rotterdam, 3000 CA, Neth.
SO Immunology Letters (2004), 91(2-3), 147-154
CODEN: IMLED6; ISSN: 0165-2478
PB Elsevier Science B.V.
DT Journal
LA English
RE.CNT 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
TI Evidence for local inflammation in complex regional pain syndrome type 1
AB BACKGROUND: The pathophysiol. of complex regional pain syndrome type 1 (CRPS 1) is still a matter of debate. Peripheral afferent, efferent and central mechanisms are supposed. Based on clin. signs and symptoms (e.g. edema, local temperature changes and chronic pain) local inflammation is suspected. Aim: To determine the involvement of neuropetides, cytokines and eicosanoids as locally formed mediators of inflammation. Methods: In this study, nine patients with proven CRPS 1 were included. Disease activity and impairment was determined by means of a Visual Analog Scale, the McGill Pain Questionnaire, the difference in volume and temperature between involved and uninvolved extremities, and the reduction in active range of motion of the involved extremity. Venous blood was sampled from and suction blisters made on the involved and uninvolved extremities for measurement of cytokines interleukin (IL)-6, IL-1β and tumor necrosis factor-α (TNF-α), the neuropetides NPY and CRGP, and prostaglandin E2. Results: The patients included in this study did have a moderate to serious disease activity and impairment. In plasma, no changes of mediators of inflammation were observed In blister fluid, however, significantly higher levels of IL-6 and TNF-. alpha. in the involved extremity were observed in comparison with the uninvolved extremity. Conclusions: This is the first time that involvement of mediators of inflammation in CRPS 1 has been so clearly and directly demonstrated. This observation opens new approaches for the successful use and development of immunosuppressives in CRPS 1.

AN 2002:305303 CAPLUS <<LOGINID::20070330>>
DN 137:167971
TI Evidence for local inflammation in complex regional pain syndrome type 1
AU Huygen, Frank J. P. M.; De Bruijn, Anke G. J.; De Bruin, Martha T.; Groeneweg, J. George; Klein, Jan; Zijlstra, Freek J.
CS Pain Treatment Centre, Erasmus Medical Centre, Rotterdam, 3000 CA, Neth.
SO Mediators of Inflammation (2002), 11(1), 47-51
CODEN: MNFLEF; ISSN: 0962-9351
PB Taylor & Francis Ltd.
DT Journal
LA English
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
TI Increased production of nitric oxide stimulated by interferon-γ from peripheral blood monocytes in patients with complex regional pain syndrome
AB This study examines immediate nitric oxide (NO) release from monocytes following interleukin-1β (IL-1β), interferon-γ (IFN-γ), and tumor necrosis factor-α (TNF-. alpha.) challenge in patients with complex regional pain syndrome (CRPS). Study patients exhibited the following: (1), mech. allodynia; (2), evidence of either vasomotor or sudomotor disturbance; and (3), concordant painful allodynia documented with quant. sensory testing that was temporarily abolished with sympathetic block. Ten subjects (CRPS, N=5; control, N=5) were enrolled. Peripheral blood monocytes were challenged with 100 μL of IL-1β (1 ng), IFN-γ (1 ng), TNF-α (0.01 ng), and normal saline (NS) and the resultant immediate NO release measured. Subjects with CRPS exhibited a statistically significant increase in NO release in response to IFN-γ compared with controls. The NO responses to IFN-γ in excess of NS and as the ratio IFN-γ/NS were also significantly increased.
AN 2002:212993 CAPLUS <<LOGINID::20070330>>
DN 136:368210
TI Increased production of nitric oxide stimulated by interferon-γ from peripheral blood monocytes in patients with complex regional pain syndrome
AU Hartrick, Craig T.
CS Department of Anesthesiology and Perioperative Medicine, William Beaumont Hospital, Royal Oak, MI, 48073, USA
SO Neuroscience Letters (2002), 323(1), 75-77
CODEN: NELED5; ISSN: 0304-3940
PB Elsevier Science Ireland Ltd.
DT Journal
LA English
RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s l8 and (inhibitor or antagonist)
535258 INHIBITOR
167898 ANTAGONIST
L10 8 L8 AND (INHIBITOR OR ANTAGONIST)

=> d l10 1-8 ti

L10 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
TI Multiplex bead array assay for detection of 25 soluble cytokines in blister fluid of patients with complex regional pain syndrome type 1

L10 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
TI Hydantoin derivatives as matrix metalloprotease inhibitors and their preparation, pharmaceutical compositions, and use for the treatment of inflammatory disorders

L10 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
TI Preparation of tartaric acid functional compounds for the treatment of inflammatory disorders

L10 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
TI Multilevel pain gate model-based method for treatment of acute and persistent pain

L10 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
TI Method of biochemical treatment of persistent pain by inhibiting biochemical mediators of inflammation

L10 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
TI Methods of using and compositions comprising selective cytokine inhibitory drug for treatment, modification and management of pain

L10 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
TI Preparation of 2-(fluoroalkoxyphenylalkyl)-1,3-dihydroisoindolones as PDE4, TNF-α , and/or MMP inhibitors

L10 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
TI Method of treatment of persistent pain by inhibiting mediators of inflammation

=> d l10 2 3 5 6 7 8 ti abs bib

L10 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
TI Hydantoin derivatives as matrix metalloprotease inhibitors and their preparation, pharmaceutical compositions, and use for the treatment of inflammatory disorders
GI

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

AB This invention relates to compds. of formula I or a pharmaceutically acceptable salt, solvate or isomer thereof, which can be useful for the treatment of diseases or conditions mediated by MMPs, ADAMs, TACE, TNF-α or combinations thereof. Compds. of formula I, wherein X is S, C(R4)2, or NR4; T is H (with U and V being absent), (un)substituted alkyl, alkenyl, (un)fused (hetero)aryl, (un)fused (hetero)cyclyl, alkylaryl, or arylalkyl; U is absent or NR4, NR4C(R4)2, NR4CO, O, NR4SO2, NR4CONR4, or NR4CSNR4; V is absent or (un)substituted alkyl, (un)substituted (un)fused (hetero)aryl, (un)substituted (un)fused heterocyclyl, or (un)substituted (un)fused cycloalkyl; Y and Z are independently absent or (C(R4)2)n, NR4, CONR4, NR4CO, NR4CONR4, SO2NR4, NR4SO2, O, S, CO, SO, or SO2; n is 1 to 3; R1 and R2 are independently H, OR4, halo, (un)substituted alkyl, (un)substituted fluoroalkyl, (un)substituted (alkyl)(hetero)aryl, (un)substituted heterocyclyl, or (un)substituted arylalkyl; each R4 is independently H or alkyl; and their pharmaceutically acceptable salts, solvates, or isomer thereof are claimed in this invention. Example compound II was prepared by amination of Me 2-bromomethyl-4-methoxybenzoate with 5-aminomethyl-5-phenylhydantoin to give 5-[[(2-methoxycarbonyl-5-methoxybenzyl)amino]methyl]-5-phenylhydantoin, which underwent cyclization to give example compound II. All the invention compds. were evaluated for their inhibitory activity of

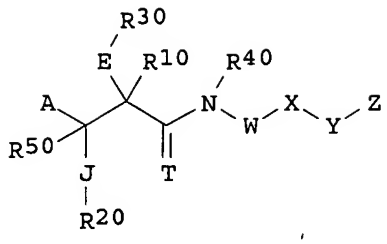
matrix metalloproteinases (MMP), a disintegrin and metalloproteases (ADAMs) and/or tumor necrosis factor α converting enzyme (TACE), and in so doing prevent the release of tumor necrosis factor α (TNF-α). The invention compds. showed inhibitory activity (Ki values) and were designated A (< 10 nM); B (10 to 100 nM); C (100 to 1000 nM); and D (> 1000 nM). For example, invention compound III showed a TACE inhibitory activity (Ki value) of 0.11 nM.

AN 2006:167381 CAPLUS <<LOGINID::20070330>>
DN 144:254128
TI Hydantoin derivatives as matrix metalloprotease inhibitors and their preparation, pharmaceutical compositions, and use for the treatment of inflammatory disorders
IN Yu, Wensheng; Tong, Ling; Chen, Lei; Kozlowski, Joseph A.; Lavey, Brian J.; Shih, Neng-Yang; Madison, Vincent S.; Zhou, Gouwei; Orth, Peter; Guo, Zhuyan; Wong, Michael K. C.; Yang, De-Yi; Kim, Seong Heon; Shankar, Bandarpalle
PA Schering Corporation, USA
SO PCT Int. Appl., 218 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

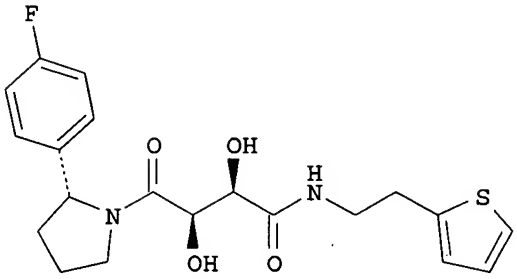
	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2006019768	A1	20060223	WO 2005-US24771	20050713
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	AU 2005275213	A1	20060223	AU 2005-275213	20050713
	CA 2573764	A1	20060223	CA 2005-2573764	20050713
PRAI	US 2004-588502P	P	20040716		
	WO 2005-US24771	W	20050713		

OS MARPAT 144:254128
RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
TI Preparation of tartaric acid functional compounds for the treatment of inflammatory disorders
GI



I



II

AB The title compds. I [A = (un)substituted benzimidazol-2-yl, imidazol-2-yl, CONH2, CSNH2; J, E = O, S, NR5 (wherein R5 = H, alkyl, alkylaryl); T = O, S; R10, R20 = H, alkyl, fluoroalkyl; R30 = H, alkyl or R30 and R40, taken together with N to which R40 is attached, are joined to form 4-7 membered (un)substituted heterocyclyl; R40, R50 = H, alkyl; W = [C(R13)2]n (wherein n = 0-5; R13 = H, halo, OH, etc.); X = a bond, alkyl, cycloalkyl, etc.; Y = a bond, O, S, NH, etc.; Z = H, alkyl, aryl, etc.; or their pharmaceutically acceptable salts] which can be useful for the treatment of diseases or conditions mediated by MMPs, ADAMs, TACE, TNF-. alpha. or combinations thereof, were prepared E.g., a multi-step synthesis of II, starting from 2,2-dimethyl-[1,3]dioxolane-4R,5R-dicarboxylic acid monomethyl ester and 2-(thien-1-yl)ethylamine, was given. The compds. I were tested against TACE (biol. data given for representative compds. I).

AN 2005:1331127 CAPLUS <<LOGINID::20070330>>

DN 144:69727

TI Preparation of tartaric acid functional compounds for the treatment of inflammatory disorders

IN Guo, Zhuyan; Orth, Peter; Zhu, Zhaoning; Mazzola, Robert D.; Chan, Tin Yau; Vaccaro, Henry A.; McKittrick, Brian; Kozlowski, Joseph A.; Lavey, Brian J.; Zhou, Guowei; Paliwal, Sunil; Wong, Shing-Chun; Shih, Neng-Yang; Ting, Pauline C.; Rosner, Kristin E.; Shipps, Gerald W., Jr.; Siddiqui, M. Arshad; Belanger, David B.; Dai, Chaoyang; Li, Dansu; Girijavallabhan, Vinay M.; Popovici-Muller, Janeta; Yu, Wensheng; Zhao, Lianyun

PA Schering Corporation, USA

SO PCT Int. Appl., 889 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005121130	A2	20051222	WO 2005-US19131	20050601
	WO 2005121130	A3	20060720		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU,

ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

	AU 2005252201	A1	20051222	AU 2005-252201	20050601
	CA 2569111	A1	20051222	CA 2005-2569111	20050601
PRAI	US 2004-576153P	P	20040602		
	WO 2005-US19131	W	20050601		

OS MARPAT 144:69727

L10 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN

TI Method of biochemical treatment of persistent pain by inhibiting biochemical mediators of inflammation

AB The invention discloses a method for the biochem. treatment of persistent pain disorders by inhibiting the biochem. mediators of inflammation in a subject, comprising administering to the subject any one of several combinations of components that are inhibitors of biochem. mediators of inflammation. The process for biochem. treatment of persistent pain disorders is based on Sota Omoigui's Law, which states: 'The origin of all pain is inflammation and the inflammatory response'. Sota Omoigui's Law of Pain unifies all pain syndromes as sharing a common origin of inflammation and the inflammatory response. The various biochem. mediators of inflammation are present in differing amts. in all pain syndromes and are responsible for the pain experience. Classification and treatment of pain syndromes should depend on the complex inflammatory profile. A variety of mediators are generated by tissue injury and inflammation. These include substances produced by damaged tissue, substances of vascular origin as well as substances released by nerve fibers themselves, sympathetic fibers and various immune cells. Biochem. mediators of inflammation that are targeted for inhibition include but are not limited to: prostaglandin, nitric oxide, tumor necrosis factor α, interleukin 1α, interleukin 1β, interleukin 4, Interleukin 6, and interleukin 8, histamine and serotonin, substance P, matrix metalloproteinase, calcitonin gene-related peptide, vasoactive intestinal peptide, as well as the potent inflammatory mediator peptide proteins neurokinin A, bradykinin, kallidin and T-kinin.

AN 2005:611671 CAPLUS <<LOGINID::20070330>>

DN 143:126805

TI Method of biochemical treatment of persistent pain by inhibiting biochemical mediators of inflammation

IN Omoigui, Osemwota Sota

PA USA

SO U.S. Pat. Appl. Publ., 40 pp., Cont.-in-part of U.S. Ser. No. 224,743.
CODEN: USXXCO

DT Patent

LA English

FAN.CNT 6

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2005152905	A1	20050714	US 2005-58371	20050216
	US 2004038874	A1	20040226	US 2002-224743	20020822
	US 2006275294	A1	20061207	US 2006-279239	20060410
PRAI	US 2002-224743	A2	20020822		
	US 2004-961037	A2	20041012		
	US 2005-58371	A2	20050216		
	US 2005-122030	A2	20050505		
	US 2005-268609	A2	20051108		

L10 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN

TI Methods of using and compositions comprising selective cytokine inhibitory drug for treatment, modification and management of pain

AB Methods of treating, preventing, modifying and managing various types of

pain are disclosed. Specific methods comprise the administration of a selective cytokine inhibitory drug, or a pharmaceutically acceptable salt, solvate, hydrate, stereoisomer, clathrate, or prodrug thereof, alone or in combination with a second active agent and/or surgery, psychol. or phys. therapy. Pharmaceutical compns., single unit dosage forms, and kits suitable for use in methods of the invention are also disclosed.

AN 2005:426388 CAPLUS <<LOGINID::20070330>>

DN 142:457121

TI Methods of using and compositions comprising selective cytokine inhibitory drug for treatment, modification and management of pain

IN Zeldis, Jerome B.; Faleck, Herbert; Manning, Donald C.

PA Celgene Corporation, USA

SO PCT Int. Appl., 85 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 5

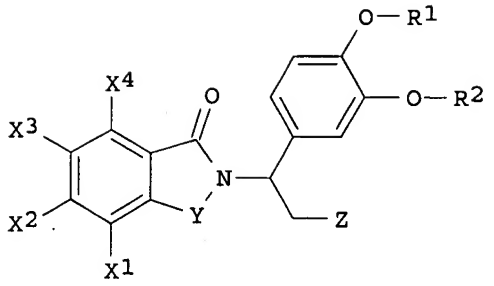
	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005043971	A2	20050519	WO 2004-US12722	20040423
	WO 2005043971	A3	20050714		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	US 2005203142	A1	20050915	US 2003-693794	20031023
	AU 2004286819	A1	20050519	AU 2004-286819	20040423
	CA 2543132	A1	20050519	CA 2004-2543132	20040423
	EP 1679967	A2	20060719	EP 2004-750613	20040423
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR				
	BR 2004015649	A	20061219	BR 2004-15649	20040423
	CN 1897816	A	20070117	CN 2004-80038252	20040423
PRAI	US 2003-693794	A	20031023		
	US 2002-421003P	P	20021024		
	US 2003-693722	A	20031023		
	WO 2004-US12722	W	20040423		

OS MARPAT 142:457121

L10 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN

TI Preparation of 2-(fluoroalkoxyphenylalkyl)-1,3-dihydroisoindolones as PDE4, TNF-α , and/or MMP inhibitors

GI



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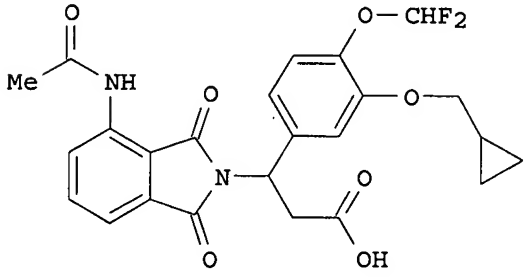

II

AB Title compds. I [wherein X1-X4 = independently H, halo, NO2, NH2, CF3, alkyl, cycloalkyl(alkyl), NR7R8-(alkyl), R8CONH-(alkyl), NR7R8CONH-(alkyl), R8OCONH-(alkyl), R8O-(alkyl), imidazolyl(alkyl), pyrrolyl(alkyl), oxadiazolyl(alkyl), triazolyl(alkyl); or X1 and X2 or X2 and X3 or X3 and X4 may be taken together to form a (hetero)cycloalkyl ring; Y = CO, CH2, CH2CO, COCH2, SO2; Z = H, COR3, alkylsulfonyl(alkyl), alkyl, CH2OH, alkoxymethyl, CN; R1 and R2 = independently CHF2, alkyl, cycloalkyl(alkyl); at least one of R1 and R2 = CHF2; R3 = NR4R5, alkyl, OH, alkoxy, (un)substituted Ph, PhCH2; R4 and R5 = independently H, alkyl, OH, OCOR6; R6 = alkyl(amino), Ph, PhCH2, aryl; R7 and R8 = independently H, alkyl, cycloalkyl(alkyl), NR7R8-alkyl, R8O-alkyl, Ph, PhCH2, aryl; or pharmaceutically acceptable salts, hydrates, solvates, clathrates, stereoisomers, and prodrugs thereof] were prepared For example, alkylation of 3,4-dihydroxybenzaldehyde with chlorodifluoromethane in the presence of K2CO3 in DMF gave 4-difluoromethoxy-3-hydroxybenzaldehyde (15%), which was further alkylated with bromomethylcyclopropane under the same conditions to afford 3-cyclopropylmethoxy-4-difluoromethoxybenzaldehyde (100%). Reaction of the benzaldehyde with ammonium acetate in 95% EtOH, followed by addition of malonic acid provided 3-amino-3-(3-cyclopropylmethoxy-4-difluoromethoxyphenyl)propionic acid (52%). Condensation of the amine with 3-acetamidophthalic anhydride using sodium acetate in AcOH yielded the isoindoledione II (85%). I and their pharmaceutical compns., optionally in combination with another therapeutic agent, are useful for the treatment or prevention of diseases associated with phosphodiesterase 4 (PDE4) inhibition, abnormal tumor necrosis factor α (TNF-. alpha.) levels , and/or matrix metalloproteinase (MMP) inhibition, such as myelodysplastic syndrome, myeloproliferative disease, complex regional pain syndrome, cancer, inflammatory diseases, and autoimmune diseases (no data).

AN 2004:589381 CAPLUS <<LOGINID::20070330>>

DN 141:140314

TI Preparation of 2-(fluoroalkoxyphenylalkyl)-1,3-dihydroisoindolones as PDE4, TNF-α , and/or MMP inhibitors

IN Muller, George W.; Man, Hon-Wah; Zhang, Weihong

PA Celgene Corporation, USA

SO PCT Int. Appl., 98 pp.
CODEN: PIXXD2

DT Patent

LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004060313	A2	20040722	WO 2003-US41568	20031229
	WO 2004060313	A3	20050915		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2511843	A1	20040722	CA 2003-2511843	20031229
	AU 2003303511	A1	20040729	AU 2003-303511	20031229
	US 2004204448	A1	20041014	US 2003-748085	20031229
	US 7173058	B2	20070206		
	EP 1587474	A2	20051026	EP 2003-808605	20031229
	EP 1587474	A3	20051102		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	BR 2003017885	A	20051206	BR 2003-17885	20031229
	JP 2006515310	T	20060525	JP 2004-565816	20031229
	CN 1802353	A	20060712	CN 2003-80109907	20031229
	US 2007072902	A1	20070329	US 2006-601355	20061116
PRAI	US 2002-436975P	P	20021230		
	US 2003-748085	A3	20031229		
	WO 2003-US41568	W	20031229		

OS MARPAT 141:140314

L10 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
TI Method of treatment of persistent pain by inhibiting mediators of inflammation
AB This invention relates to a method for treating persistent pain disorders by inhibiting the biochem. mediators of inflammation in a subject comprising administering to said subject a therapeutically effective dosage of said inhibitor. Said process for treating persistent pain disorders is based on Sota Omoigui's Law, which states: The origin of all pain is inflammation and the inflammatory response. Biochem. mediators of inflammation that are targeted for inhibition include but are not limited to: prostaglandin, nitric oxide, tumor necrosis factor alpha, interleukin 1-alpha, interleukin 1-beta, interleukin-4, Interleukin-6 and interleukin-8, histamine and serotonin, substance P, Matrix Metallo-Proteinase, calcitonin gene-related peptide, vasoactive intestinal peptide as well as the potent inflammatory mediator peptide proteins neurokinin A, bradykinin, kallidin and T-kinin.
AN 2004:162447 CAPLUS <<LOGINID::20070330>>
DN 140:193061
TI Method of treatment of persistent pain by inhibiting mediators of inflammation
IN Omoigui, Osemwota
PA USA
SO U.S. Pat. Appl. Publ., 14 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 6

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004038874	A1	20040226	US 2002-224743	20020822
	US 2005152905	A1	20050714	US 2005-58371	20050216

	US 2006275294	A1	20061207	US 2006-279239	20060410
PRAI	US 2002-224743	A2	20020822		
	US 2004-961037	A2	20041012		
	US 2005-58371	A2	20050216		
	US 2005-122030	A2	20050505		
	US 2005-268609	A2	20051108		